# Closing Remarks: Environmental Health in the Twenty-First Century

### by Bernard D. Goldstein\*

#### Introduction

It is a challenge to summarize a meeting of this nature. The presentations have been interesting and provocative. Some have looked forward, others have simply tried to take the next step, while still others have actually gone backwards to recount for us pertinent lessons of the past. The bulk of the presentations have discussed industries or agents. I will not try to recapitulate all of these interesting presentations, rather I will attempt to cross-cut by starting from the health problems and diseases caused by environmental agents.

#### The Only Certain Prediction

While many of the predictions we have heard at this conference have a high likelihood, there is only one prediction that can be made with certainty. In the twenty-first century there will be a significant environmental health problem that no one in the twentieth century will have predicted. The vagaries of human existence and the ingenuity of the human mind will certainly lead to unforeseen environmental health hazards. There is an important implication of this certainty. We need a healthy science base to anticipate as many of the changes as possible, and we should be responsive to those problems that we fail to predict until some adverse effect on human health or the environment has occurred. Only through the support of basic mechanistic research will we have a responsive scientific base to be able to deal with such problems. Quite obviously, a purely applied approach to environmental health problems will leave us always chasing after the next unknown much too late.

## **Environmental Causes of Human Diseases**

The good news is that we will learn more about the environmental causes of human diseases. The reason this is good news is that environmental causes are preventable causes. Rather than discussing cancer and

organ systems such as the kidney, for which there is already excellent evidence of a role for environmental factors in disease causation, let me make this point by turning to the area of rheumatology. This subspecialty field deals primarily with an organ system responsible for a large percentage of crippling diseases as well as some that are potentially fatal such as systemic lupus erythematosus (SLE). Rheumatoid arthritis, SLE, and related collagen-vascular disorders appear to have major immunologic components in their pathophysiology. It has long been recognized that an SLE-like disease can be provoked by various therapeutic agents (1). Similarly, other autoimmune phenomena in which the immune system attacks components of the body also have been demonstrated to be provoked by drugs. An excellent example is the antihypertensive drug  $\alpha$ -methyldopa (Aldomet) (2). Approximately 15% of individuals receiving this drug develop discernible antibodies to their own red blood cells. Of note is that of the individuals who do develop these antibodies, only a small percentage also develop hemolysis, which is the destruction of the red blood cells. Furthermore, there is not an obvious one-toone relationship between the time of drug administration and the hemolysis. Cessation of therapy does not lead to an immediate response, but rather a slow decline in the extent of hemolysis or detectable presence of antibody. Were it not for the fact that we know that we are giving this therapeutic chemical to large numbers of people who have similar manifestations, we would not be able to detect the linkage of the chemical to the disease.

My contention is that we will find that environmental chemicals are also responsible for autoimmune phenomena at least partially underlying rheumatoid arthritis and SLE. This is supported by the fact that relatives of those affected have a greater likelihood of having similar serum abnormalities, such as autoantibodies, than does the general population; for SLE it has been shown that it is more likely for a house occupant, such as a spouse, to have a similar antibody than for a blood relative who does not live in the same home. While this has led those studying the phenomenon to hypothesize that some viral infectious agent is involved, in my judgment it is far more likely to be due to a common chemical under the kitchen sink or elsewhere in the home.

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The findings with  $\alpha$ -methyldopa also strongly suggest that there is some inherited component to this responsiveness; otherwise why do only a certain percentage of individuals develop the antibodies and only a small percentage of those who develop the antibodies actually get the disease? This leads to another prediction. By the twenty-first century we will develop an understanding of the interface between environment and heredity, which will allow us to comprehend the role of inheritance in the differing reactions of individuals to chemical and physical agents. We should emphasize that the development of accurate markers of human susceptibility will bring with it some very important ethical questions that need to be addressed (3).

#### The Environment and Aging

The aging of our population is a very important trend with many societal implications. The number of individuals in their eighth and ninth decade is increasing dramatically. A healthy life style and a healthy environment will be important factors in quality of life considerations among the aged population. This will put a premium in the twenty-first century on investigating the mechanisms of aging and in determining means for the healthy aged to have an improved quality of life.

The mechanism of the subtle changes in structure and function that occur under the name of aging remains a scientific puzzle. It is not unreasonable to predict that in the twenty-first century we will come to recognize more and more a role for environmental and chemical and physical factors in the aging process. For example, it is quite conceivable that repetitive exposure to relatively low levels of air pollutants will affect the lung function of an 80 year old to an extent that it will become a limiting factor in daily activities.

#### **Risk Communication**

The effective prevention of environmentally caused diseases requires an ability to communicate the extent of risk to society. Decision-making in the area of environmental health risk always carries some degree of trade-off. It is unreasonable to expect that this trade-off will be performed optimally if the extent and character of the risk is not well understood by those involved in making decisions and by those who will be affected by the decision.

Research resulting in improved understanding of risk is a necessary prerequisite for effective communication of the risk. In addition, basic research should be effective in not only understanding the risk but also in directly leading to better ways to communicate the risk. There are many examples of increased effectiveness of public health measures simply because of the development of mechanistic understanding of toxicity leading to better surveillance techniques. For example, the zinc protoporphyrin screening test for lead poisoning, which has greatly increased the likelihood that children and

families will be rapidly and effectively screened, developed out of basic advances in the understanding of heme synthesis and the role of metals in the fluorescence of protophophyrin (4).

As another example, consider the case of radon. Based on the relative lack of public response, there is no question that we have done a poor job of communicating the risks of radon. Despite a variety of efforts to alert the public to these risks, very few individuals have had their homes checked, even in high-risk areas. One needed research approach is to devise a relatively simple tool to follow radon progeny within the lung. While this is an important technique in understanding the basic mechanisms of toxicity of radon, it could also be a very powerful risk communication tool. Imagine a device hooked up with bells and whistles, much like a Geiger counter, that would provide relatively rapid response and be portable as well. Compare the impact of finding high radon on a device such as this used on your children's lungs after they have been playing in the basement, as compared to the current approach of waiting for the results from a canister left in your basement for a few weeks.

#### **Societal Changes**

Perhaps the most difficult challenge to a speaker summarizing this session is to say something original about the impact of societal trends on environmental health in the twenty-first century. Fortunately, my job has been made much easier by today's news. Many of you. I am sure, have seen the television advertisements placed by a major airline celebrating the banning of smoking on its flights. Apparently these were developed by the same advertising firm that also has an \$84 million account with RJR-Nabisco Corporation. RJR-Nabisco is an outgrowth of the Reynolds' tobacco interests buying up the National Biscuit Company, the maker of such relatively innocuous products as Ritz Crackers and Fig Newtons. The trend to corporate mergers and takeovers leads to the worrisome possibility that major corporations whose products or activities are of adverse consequences to environmental health will attempt to protect their interests by developing increased economic leverage through the buy-out of corporations that are relatively neutral in terms of public health. The recent report that RJR-Nabisco is firing their ad agency because it made an anti-smoking advertisement is a clear example of the use of naked economic power to protect that portion of a corporation that has participated in the poisoning and death of literally millions of Americans with billions of dollars of economic consequences.

There is one aspect of this situation that perhaps RJR-Nabisco has not thought of: the same approach that gives lateral economic strength to the corporation also puts a counter-weapon in the hands of those who oppose the corporation's practices. Using the same reasoning that appears to have motivated RJR-Nabisco in boycotting their advertising agency, it will be possible for

citizen's groups to attack the harmful aspects of the corporation by boycotting its more neutral consumer products. I, for one, would strongly advocate a boycott of Fig Newtons, which have been my favorite since childhood. I expect that as we continue to see further takeovers and concentration of economic power within a few corporations, the twenty-first century will see more and more of a use of selective economic boycotts by consumer and health advocate groups.

#### **Summary and Conclusions**

I began this presentation by stating that there was only one certainty: something unpredicted will occur. There is actually another certainty; we have all participated in what will be a major academic and governmental growth industry in the next 12 years, the holding of conferences predicting the twenty-first century. Let us remember that Dr. David Rall and his staff were the first. As a hallmark of NIEHS, they have shown that

they have the foresight to make the necessary plans for the future. It is clear that we can look forward to some exciting research as we move into the twenty-first century. This specific excitement should be more then met by a sense of accomplishment as the data that are obtained are used to prevent disease.

#### REFERENCES

- Mansilla-Tinoco, R., Harland, S. J., Ryan, P. J., Bernstein, R. M., Dollery, C. T., Hughes, G. R. V., Bulpitt, C. J., Morgan, A., and Jones, J. M. Hydralazine anti-nuclear antibodies, and the lupus syndrome. Br. Med. J. 284: 936-939 (1982).
- Worlledge, S. M. Immune drug-induced hemolytic anemias. Semin. Hematol. 10: 327 (1973).
- Biological Markers in Environmental Health Research. Environ. Health Perspect. 74: 3-9 (1987).
- Lamola, A. A., Piomelli, S., Poh-Fitzpatrick, M. B., Yamane, T., and Harber, L. C. Erythropoietic protoporphyria and lead intoxication: the molecular basis for difference in cutaneous photosensitivity. II. Different binding of erythrocyte protoporphyrin to hemoglobin. J. Clin. Invest. 56: 1528-1535 (1975).